Acute mediastinitis

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Acute bacterial infection of the mediastinum can evoke a devastating disease which, in its fulminating form, is often unresponsive to the best therapeutic efforts. However, if mediastinitis is diagnosed before it reaches the morbid pathological state, appropriate antibiotic therapy and well-planned surgical intervention may favorably alter the prognosis.

CASE REPORT

A 54-year-old man developed fever, a sore throat, and difficulty in swallowing. On the third day of illness, he sought medical consultation and was diagnosed with laryngitis and pending pneumonia. He was prescribed oral antibiotics and a sinus decongestant, but on the fifth day of illness, his fever spiked and he began experiencing chest discomfort. He went to the emergency department at Baylor University Medical Center and was admitted to the otolaryngology service.

At the time of admission, he was dyspneic and hoarse. On physical examination, the left pharyngeal area was erythematous and swollen, and the left side of the neck was tender and indurated. His white blood cell count was $15.3 \times 10^3/\mu L$. A chest radiograph showed no acute process. The patient was admitted to the intensive care unit and treated with intravenous cefuroxime and steroids.

On hospital day 3, he was taken to the operating room. Examination under general anesthesia revealed a left deep parapharyngeal abscess with spontaneous rupture in the pharyngeal space. The area of rupture was repaired, and the abscess was drained with a left lateral neck approach. Penrose drains were inserted, and neomycin was prescribed. Bacteriology cultures showed gram-positive anaerobic cocci and rods and *Staphylococcus haemolyticus*. Consequently, vancomycin, to which the organisms are sensitive, was prescribed in place of neomycin.

The patient's serum fibrinogen was 650 mg/dL on hospital day 3, so he was started on dextran 40 (Rheomacrodex) to decrease the viscosity and platelet margination and to avoid vascular thrombosis. However, the serum fibrinogen climbed to 1079 mg/dL. In addition to dextran 40, low-molecular-weight heparins were given subcutaneously. The serum fibrinogen continued to fluctuate between 800 and 950 mg/dL until the active pathology was arrested and the level started to decline.

Two days after the peritonsillar abscess was drained, the patient became septic. Computed tomography (CT) showed fluid collection and soft tissue induration of the right neck and anterior superior mediastinum. There was air in the soft tissues of the right neck and anterior superior mediastinum, and a pocket of air was present in the upper posterior mediastinum, with air dissection in the soft tissue of the mediastinum down to the diaphragm *(Figure 1)*. In addition, a right hydrothorax was observed.

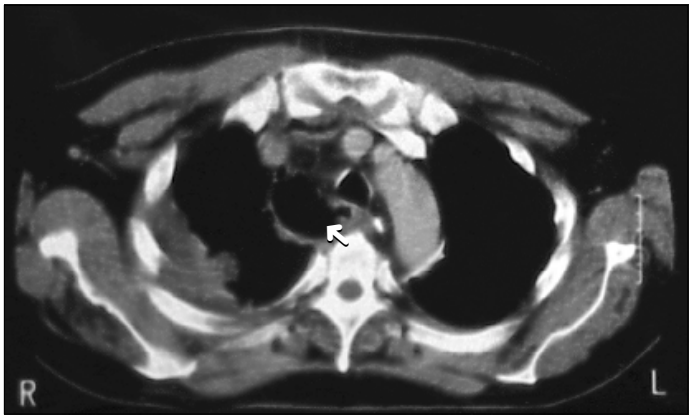

Figure 1. CT scan of chest showing air dissection in the mediastinum (arrow).

On hospital day 8, the abscess of the right neck and anterior superior mediastinum was drained through a transverse suprasternal incision. Penrose and sump drains were inserted. Through a left anterior second space incision, the large air pocket in the upper posterior mediastinum was drained extrapleurally using a chest tube with underwater seal. The hydrothorax was concomitantly drained using 2 large Argyle tubes connected to underwater seal. Pathological findings in the neck and upper mediastinum indicated pus mixed with blood and significant amounts of fasciitis and necrosis, with necrosis more preponderant than pus. The surgical planes were indurated, and the anatomic planes were effaced. A culture grew β-hemolytic streptococci and anaerobic gram-positive rods sensitive to vancomycin.

Postoperatively, the patient's temperature and white blood cell count improved. Because serum albumin was low, hyperalimentation was started. The purulent drainage continued from both the neck and superior mediastinum. A component of oral fluid was noticed in the drainage on hospital day 10, 4 days after

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drainage of the right neck and mediastinum. A breakdown in the repair of the ruptured pharyngeal mucosa that had been performed during the initial surgery was suspected. The patient became very septic. A CT scan of the neck and chest obtained on hospital day 12 showed fluid in the right side of the neck, upper anterior mediastinum, upper posterior mediastinum, empyema thoracis, and left hydrothorax.

On hospital day 13, the patient's oral cavity was examined, which confirmed the breakdown of the suture line overlying the abscess area. The abscess cavity was packed with iodoform gauze to divert oral fluid from reaching the infected spaces in the right neck and anterior mediastinum. When the latter spaces were reexplored, purulent fluid was encountered and drained; the area was then irrigated with antibiotic solution. A new surge of granulation tissues was also observed, and new sump drains were inserted. The patient was then repositioned in the left lateral decubitus position. Right posterolateral muscle-sparing thoracotomy was performed. The abscess in the upper posterior mediastinum was partially resected, and its purulent contents and necrotic tissues were removed. Again, the necrotic tissues were preponderant compared with the amount of pus. Decortication of the lung was carried out. The pleural space was drained with 3 chest tubes connected to underwater seal, and the left hydrothorax was drained with 2 chest tubes connected to underwater seal. A culture grew β-hemolytic streptococci, anaerobic gram-positive cocci, and *Staphylococcus haemolyticus*. Imipenum-cilastatin sodium (Primaxin) was given in addition to vancomycin.

Postoperatively, the patient slowly improved. However, serosanguineous drainage from the right chest continued, possibly due to hypoalbuminemia. The patient's caloric intake was increased. On hospital day 21, right chest tubes started draining purulent fluid, which cleared up after 2 days. A CT scan was obtained on hospital day 25. It showed loculation of fluid in the right chest and empyema thoracis on the left, despite patency of chest tubes bilaterally. On hospital day 26, the patient underwent thoracoscopic evacuation of loculations of the right hemithorax. Concomitantly, he had decortication of the left lung and evacuation of the empyema. New chest tubes were inserted and connected to underwater seal. A culture grew *Mycobacterium avium complex*. Because of this finding, at a later date the patient was checked for HIV, but the results were negative. On the third postoperative day, the serous drainage from the left chest tubes began to acquire thick, fibrinous components, and loculation began to show in the left hemithorax. The patient was started on streptokinase installation into the left thoracic cavity with a daily dose of 250,000 IU. The chest tubes were clamped for 2 hours and then returned to suction. Abundant fibrinous materials started to drain through the chest tubes. Loculations disappeared in about 3 days. Thereafter, the drainage continued to be clear until the chest tubes were removed.

On discharge, the serum fibrinogen level was 600 mg/dL. The patient was continued on low-molecular-weight heparins for 2 more weeks until the level returned to normal.

DISCUSSION

Acute mediastinitis, an infection that has become uncommon since the advent of effective antibiotics, can be primary or secondary.

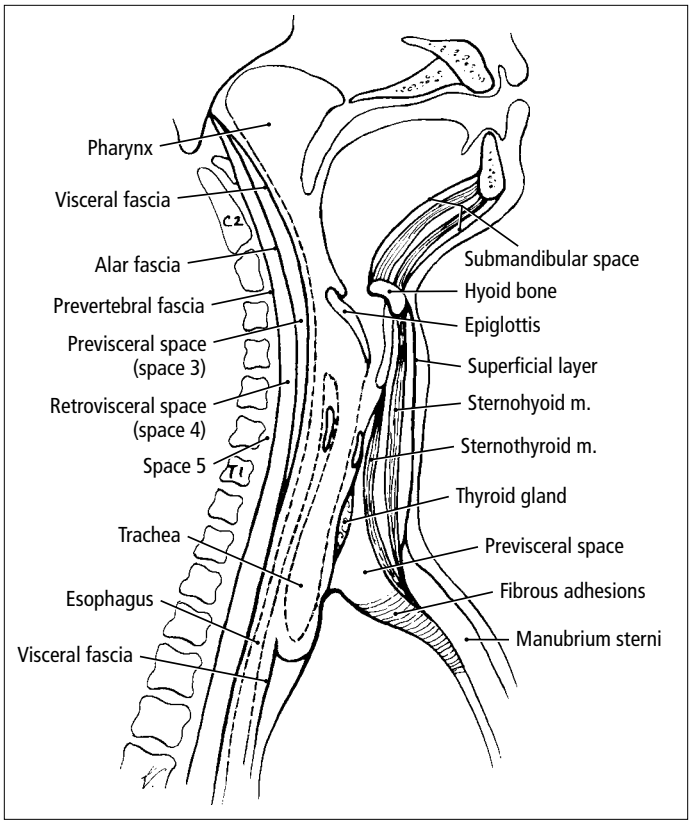

Figure 2. Diagram of fasciae and spaces of the head, neck, and mediastinum in the midsagittal section. From Grodinsky M, Holyoke EA. The fasciae and fascial spaces of the head, neck and adjacent regions. *American Journal of Anatomy* 1938;63:367–408. Copyright © John Wiley & Sons, Inc. Reprinted by permission of John Wiley & Sons, Inc.

Primary cases of mediastinitis are rare (1). They can occur spontaneously or in connection with epiglottitis, pharyngitis, pneumonia, pericarditis, and bronchitis (2–6). Although the infection may be self-limiting and completely resolve, it may also spread into the neck or into the broad ligament of the lung (2).

The great majority of mediastinal infections are secondary, originating from many sources. Most secondary mediastinal infections are related to esophageal disruption, although not all esophageal perforations lead to mediastinitis. Puncture perforations tend to heal spontaneously, while large tears require surgery (1). In esophageal erosion caused by the presence of a foreign body, an inflammatory reaction occurs and invasive mediastinitis does not develop (7).

Deep sternotomy wound infection is another source of secondary mediastinitis. The sternotomy incision has been widely used in open intracardiac procedures since it was proposed by Julian in 1957 (8). McClelland reviewed the incidence of deep sternotomy wound infection and mediastinitis between 1984 and 1996, finding a rate of 0.3% to 5% (9). One group of patients in this review had a 0.46% incidence of deep sternotomy wound infections and an 11% mortality rate.

Respiratory tract infections are rarely the cause of mediastinitis, due to antibiotics and improved oral hygiene (10). However, oropharyngeal infections, such as quinsy, Ludwig's angina, and retropharyngeal abscess, are cause for concern since they tend to spread along the fascial planes. These infections can cause morbid necrotizing mediastinitis.

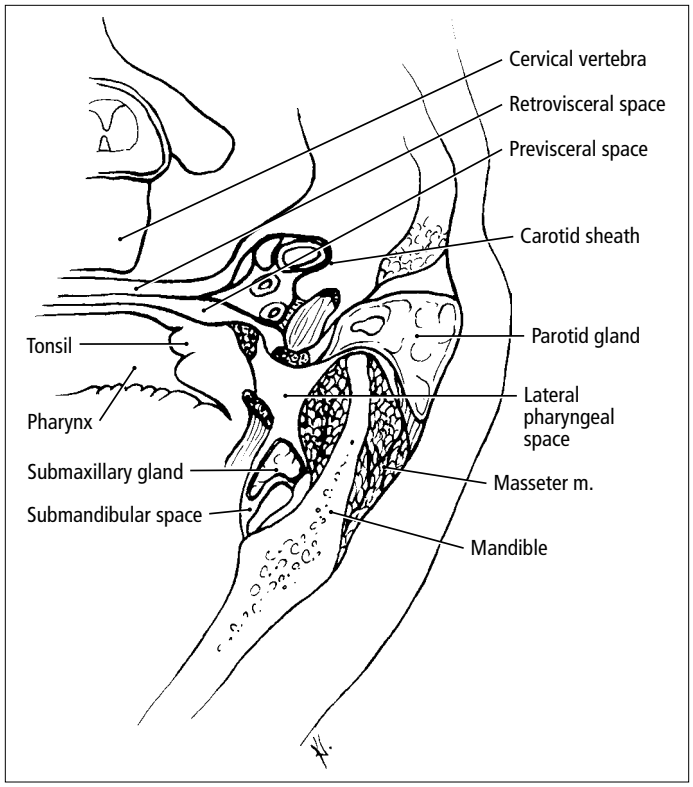

Figure 3. Transverse section through the tongue and the palatine tonsil. From Grodinsky M, Holyoke EA. The fasciae and fascial spaces of the head, neck and adjacent regions. *American Journal of Anatomy* 1938;63:367–408. Copyright © John Wiley & Sons, Inc. Reprinted by permission of John Wiley & Sons, Inc.

At this time, metastatic infections, retroperitoneal and subphrenic infections, and osteomyelitis of the spine and ribs seldom lead to mediastinitis. However, other new sources for infection are developing. AIDS and immunosuppression are leading to new combinations of infectious processes.

It is important to understand the anatomy of the fascial spaces connecting the neck and pharynx to the mediastinum since infection should be intercepted and drained there. Certain virulent infections can freely spread through fascial planes and along fascial spaces (11) *(Figure 2)*. The fascial planes can influence the early spread of mediastinitis and are key to understanding the symptoms and planning treatment (10).

The lateral pharyngeal space is a transfer point for infections originating in the mandible, parotid gland, tonsils, and cellulitis of the sublingual and submaxillary spaces *(Figure 3)*. Such infections can move through the lateral pharyngeal space to the connecting previsceral space. Infections in the previsceral space may reach the mediastinum by spreading through the neck or breaking through the alar fascia to reach the danger space (the retrovisceral space).

Respiratory dynamics influence the spread of infection along these spaces. Fluctuation in the negative intrathoracic pressures tends to draw the contents of the fascial spaces into the mediastinum. Oral contents such as air, saliva, and microorganisms are sucked into the mediastinum, contributing to virulent necrotizing mediastinitis (12).

Postoperative pain in these gravely ill patients must be controlled to prevent pneumonia and postsurgical pulmonary atelectasis. Effective pain relief can be provided through epidural analgesia or paravertebral block, along with morphine sulfate or meperidine hydrochloride (Demerol) given via patient-controlled pump. Intravenous ketorolac tromethamine (Toradol) is also effective for pain relief.

It is essential to diagnose acute mediastinitis early to circumvent its lethal consequences. Once the symptoms are recognized, appropriate antibiotics can be administered and surgery can be planned.

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